‘DOES BLADDER OUTLET OBSTRUCTION PREDISPOSE TO ABDOMINAL STRAINING IN MEN?’

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A dissertation submitted to The Dr. M.G.R. Medical University, Tamilnadu, in partial fulfillment of the requirements for M.Ch. Branch-IV (Genitourinary surgery) examination to be held in August 2009.

CERTIFICATE

This is to certify that this dissertation entitled **'DOES BLADDER OUTLET OBSTRUCTION PREDISPOSE TO ABDOMINAL STRAINING IN MEN?'** is bonafide work done by **Dr.Sameer Grover** in partial fulfillment of the rules and regulation for M.Ch. Br. IV (Genitourinary Surgery) examination of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, to be held in August 2009.

Dr. Nitin Kekre M.S, DNB (Urology)
Professor & Head,
Dept. of Urology,
Christian Medical College & Hospital,
Vellore.

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CONTENTS

INTRODUCTION

Abdominal straining is assumed to be a symptom of bladder outlet obstruction(BOO). It is included in the various symptom scores which are used for the assessment of patients presenting with lower urinary tract symptoms (LUTS) suggestive of BOO.[1-4] It is presumed that straining augments urinary flow. However there is a sparse literature linking abdominal straining and outlet obstruction. Furthermore there is little evidence that straining is specifically a feature of BOO and the effect it has on the flow of young asymptomatic men or symptomatic men. It has been shown that upto 25% of men strain habitually though no large longitudinal population based studies have been done[5]. Uroflowmetry and postvoid residual urine volume (PVR) are useful screening tests in the evaluation of men with LUTS but neither can make a definitive diagnosis of BOO. Most men with BOO have diminished flow rates, [6] and 90% of neurologically normal men with a maximum flow rate (Qmax) of

less than 10 mL/sec are obstructed.[7] conversely, 25% to 30% of men with

decreased flow are not obstructed. Decreased uroflow can result from impaired detrusor contractility or obstruction. Without the synchronous measurement of detrusor pressure (Pdet), uroflow is unable to distinguish between these 2 entities.[8-10]. Similarly, a normal uroflow does not exclude outlet obstruction. Urodynamics with pressure flow studies remain the gold standard for diagnosing BOO and other voiding and storage abnormalities responsible for LUTS and voiding dysfunction. We wished to determine if there is any correlation between the symptoms of straining to objective urodynamic parameters.

AIM OF THE STUDY

To evaluate the relationship between bladder outlet obstruction and abdominal straining in men > 45 yrs of age.

Review of Literature

Pathophysiology of BOO: The partially obstructed urethra, detrusor muscle and the central nervous system function interact to produce lower urinary tact symptoms (LUTS). These were historically referred to as ‘prostatism’ . There are several mechanisms by which Benign prostatic hyperplasia (BPH) may cause obstruction such as a prominent median lobe acting as a ball valve, a dynamic obstruction related to the contractile properties of prostatic smooth muscle, a static obstruction resulting from an enlarged prostate enveloping the prostatic urethra, or a restricted surgical capsule. Each of these mechanisms is clinically feasible and components of each are likely to be present in most instances. The result is a raised intravesical pressure and a reduction in flow which leads to the gradual development of secondary changes in the muscle itself.

Effect of obstruction on the bladder: Gross anatomical, histological,

cellular and molecular alterations in bladder wall, which result from obstruction of the urethra impair its function and add to the symptomatology of BPH[11]. Hypertrophy of the detrusor muscle in early phases of outflow obstruction allows a compensatory increase in detrusor pressure in order to maintain flow in the presence of increased outflow resistance. With persistent obstruction however decreased compliance in the bladder wall and impaired emptying occur owing to the deposition of increasing amounts of extracellular matrix (ECM)[12]. Acute urinary retention may occur during the process and may be related to bladder failure, as well as to sudden increase in outflow obstruction. The alteration in ECM is probably the predominant pathophysiological feature in long term obstruction. Studies from the rabbit model of obstruction have shown that significant smooth muscle hyperplasia is induced when the load is increased and that this is associated with down regulation of myosin light chain (MLC) Expression. This effect contributes to the decreased smooth muscle contractility and moreover results in development of dedifferentiated smooth muscle phenotype[13]

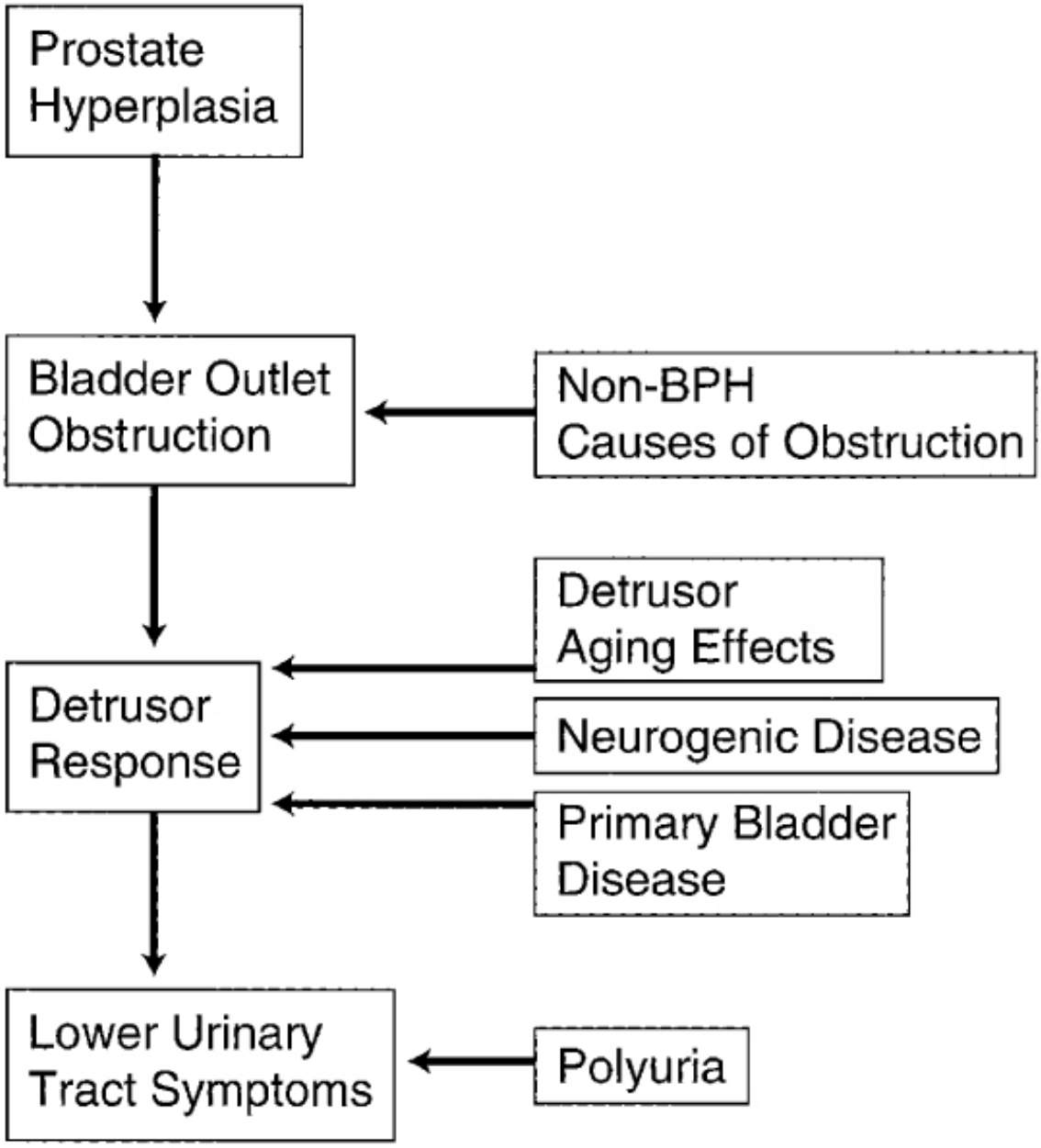

Figure 1:The pathophysiology of BOO*(Adapted from Wein: Campbell-Walsh Urology, 8th ed. Physiology and Pharmacology of the Bladder and Urethra, p1303)

Natural History of Benign Prostatic Enlargement:

The natural history of a disease refers to the progression of the untreated disease over time. Clinical endpoints of progression for BPH include the

development of more severe symptoms, bladder dysfunction manifested by incomplete emptying or detrusor instability, more severe bladder outlet obstruction, acute urinary retention (AUR), recurrent UTI, urosepsis, chronic renal insufficiency, bladder stones, incontinence, and hematuria. The natural history of BPH is incompletely understood because of the absence of a uniform definition of the disease and the lack of rigorous studies. Insights into the natural history of benign prostatic enlargement can be gleaned from the longitudinal follow-up of the Olmstead County Study of Urinary Symptoms and Health Status.[14]A relatively small subset of men between the ages of 40 and 79 were randomly selected from the Olmstead County community and underwent transrectal ultrasonography at baseline

and 6 years later. A mixed-effects regression model showed that prostate volume increased by about 1.6% per year on average.

Men with larger prostates at baseline experienced the greatest increase in prostatic volume. Jacobsen and colleagues27 reported

on LUTS progression in the Olmstead County Study over an interval of 42 months. The AUA symptom score was categorized as mild (0-7) versus

moderate to severe (8-35). There was much movement across symptom categories during the follow-up interval. At 42 months, 22% of men with mild symptoms crossed over to moderate to severe symptoms. A regression model showed that the average symptom score change over time was 0.18 symptom units per year. The AUA symptom score increased during this interval of time in all age categories. The greatest mean symptom score progression was observed in the 60- to 69-yearold

age group. The Medical Therapy of Prostatic Symptoms (MTOPS) study represents the longest placebo-controlled trial to date of men

with BPH.[15] It is important to note that prostate volume was not an inclusion criterion for enrollment. Thus, the placebo arm provides

insights into the natural history of men with moderate to severe LUTS and decreased peak urinary flow rates, which imply some level of bladder outlet obstruction. The objective of the MTOPS study was to examine the impact of medical therapies on BPH progression. In this study, BPH progression was defined as a 4-point increase in AUA symptom score or the development of

AUR, chronic renal insufficiency or socially unacceptable incontinence, or recurrent UTI or urosepsis. The final analysis of the MTOPS study was recently conducted with a mean follow up of 4.5 years. The only clinically relevant progression rates were

observed for symptom progression and AUR. The overall progression rate (events/100 patient-years) was 4.5 in the placebo group. The MTOPS study demonstrated that the development of AUR is quite common in men with clinical BPH. This is consistent

with the Olmstead County Study of Urinary Symptoms. and Health Status, which reported a cumulative incidence rate for AUR of

6.8 Per thousand person-years. With a multivariate analysis, age at baseline, symptom severity, and peak flow rate independently predicted risk of AUR. Prostate volume was not evaluable as a predictive factor as only a small subset of men underwent prostate volume determination at baseline. Based on information from the Olmstead County Study, a 60-year-old man with moderate to severe symptoms has a 13.7% chance of developing AUR by age 70. The placebo arms of long-term studies evaluating the safety and

effectiveness of the 5ARIs dutasteride [16,17] and finasteride[18] provide insights into the risk of AUR in men with LUTS, bladder outlet obstruction, and an enlarged prostate. In men with prostates over 58 cm3, the risk of AUR in the finasteride study placebo group over the 4-year period was 22%.

LUTS and BPH: Benign prostatic hyperplasia (BPH) is a progressive disease that is commonly associated with bothersome

lower urinary tract symptoms (LUTS) such as urinary frequency, urgency, nocturia, decreased and intermittent force of stream and the sensation of incomplete bladder emptying. The term 'BPH' actually refers to a histological condition, namely the presence of stromal-glandular hyperplasia within the prostate gland[19]. The condition becomes clinically relevant if and when it is associated with bothersome LUTS; however, the relationship between BPH and LUTS is complex, because not all men with histological BPH will develop significant LUTS, while other men who do not have histological BPH will develop LUTS. Benign prostatic enlargement (BPE) is another component of the LUTS /BPH constellation [20]. Reflecting the

complex relationship between age related changes in the prostate, not all men with histological BPH will develop BPE; in addition, not all men with LUTS will have concomitant BPE, and not all men with BPE will have bothersome

LUTS. The final component of this complex relationship is bladder outlet obstruction (BOO). This results from a pressure gradient at the bladder neck/ prostatic urethra and may lead to compression of the urethra, compromised urinary flow and deterioration of the upper urinary tract with renal failure. Yet again, not all men with BPH/ BPE and LUTS will have BOO, and there are causes of BOO other than BPH/ BPE (e.g. primary bladder neck sclerosis or a

urethral stricture). The causes of LUTS are multifactorial, although

BPE secondary to BPH is a major contributing factor. The prevalence of LUTS in Europe varies with age, ranging from 14% for men in their fourth decade of life to > 40% for men in their sixth decade[21]. Although bothersome LUTS are commonly the

only determinant for a BPH diagnosis in clinical practice, simple investigations exist that can be highly effective in accurately diagnosing

LUTS because of BPH. The European Association of Urology (EAU) guidelines recommend a series of initial evaluations for men with LUTS suggestive of bladder obstruction;

these include taking a clinical history, using a validated questionnaire to assess symptoms, conducting a physical examination, creatinine measurement, urinalysis, flow rates, postvoid residual (PVR) volume and serum prostate-specific antigen (PSA) measurement (particularly when a diagnosis of

prostatic carcinoma would affect the decision about which therapeutic option to use)[22]. The initial evaluations recommended by the American Urological Association (AUA) are a clinical history, use of a validated questionnaire to assess symptoms, a physical examination, urinalysis and serum PSA measurement [23]

It has been demonstrated that there is a high correlation between diagnoses using medical history, serum PSA, digital rectal examination (DRE) and International Prostate Symptom Score (IPSS) and those based on a full battery of tests including ultrasonography , uroflowmetry and urodynamics[24].

Urodynamics in Diagnosis: Urodynamic studies are the most definitive tests available to determine the etiology of voiding dysfunction and lower urinary tract symptoms. The urodynamic study can be divided into 2 parts, the filling and storage phase (cystometrogram) and the voiding phase (voiding pressure flow study). The voiding phase allows one to definitively make a diagnosis of obstruction, as detrusor pressure and urinary flow rate can be measured and outlet resistance calculated. However the filling and storage phase measured by the cystometrogram (CMG) can provide useful information in the patient in whom obstruction is suspected, for example, detrusor overactivity, or involuntary contractions, may be present (with or without obstruction) and may account for symptoms. Sensation and capacity also can be determined. Another urodynamic parameter is impaired compliance. Normally the bladder should hold increasing volumes of urine at low pressures indicating a highly compliant structure (compliance =change in volume/change in

pressure). Impaired

compliance may result from several conditions including neurogenic voiding dysfunction, radiation cystitis, tuberculosis,

and chronic bladder outlet obstruction. In the case of obstruction,

compliance appears to deteriorate as a result of high intravesical

pressure generated by bladder muscular activity opposed by inappropriately high outlet resistance. Prolonged high-storage pressures are known to be detrimental to renal function.[25] The simultaneous measurement of detrusor pressure and urinary flow

rate during voluntary voiding is one of the best ways currently available to access 2 critical parameters of bladder and outlet function: detrusor contractility (normal vs. impaired) and outlet resistance (obstructed vs. unobstructed). In general, pressure-flow

studies identify 3 fundamental voiding states:

1) Low detrusor pressure and high flow rate (unobstructed)

2) High detrusor pressure and low flow rate (obstructed)

3) Low detrusor pressure with low flow rate (poor detrusor

contractility).

It is important to understand that pressure-flow studies do not always allow for an absolute classification into one distinct category. Borderline cases exist as well as cases in which there is a combination of impaired contractility and obstruction.

Measures of Outlet Resistance and Obstruction: Attempts to mathematically define urethral resistance date back to the early 1960s.[26] Early equations calculating urethral resistance, such as

R = Pves/Q (where R = resistance, Pves = vesical pressure, and Q = flow rate), followed standard hydrodynamic formulae calculating outlet resistance. Unfortunately, these concepts failed to consider that the urethra has an active and distensible nature and is not a rigid tube. They also failed to consider the importance of bladder volume. Rigid tube hydrodynamics were abandoned in favor of more dynamic ways to analyze micturition. In 1972, Griffiths introduced Bladder Output Relation (BOR), which depicted the interrelation between bladder pressure and uroflow at a given volume.[27,28] According to the BOR, for any given bladder there is

A specific bladder output relation and the higher the bladder pressure, the lower the flow and vice versa. The BOR essentially measures the function of the bladder independent of the function of the urethra. Griffiths further defined a method to evaluate urethral resistance independent of bladder function: the urethral resistance relation (URR).[29]According to this relation, as bladder pressure rises; the flow rate will be zero until the intrinsic bladder pressure

equals the intrinsic urethral pressure. At this point flow will start

and the flow rate will rise rapidly with further increases in the intrinsic bladder pressure. If pairs of simultaneously measured values of detrusor pressure and flow rate are plotted against one another throughout the course of a micturition event, a curve is obtained that shows the resistance to flow independent of detrusor function, representing the urethral resistance relation. A change in one of these relations during micturition would not affect the curve representing the other relation but would result in the point of intersection to move along that curve. In 1979, Abrams and

Griffiths defined a simple nomogram for the diagnosis of obstruction in

males.[30] The researchers collected pressure flow data on 117 males older than age 55 years, who were evaluated for possible prostatic obstruction. By comparing pressure-flow data

between these patients and plotting the Qmax on the X axis and the

detrusor pressure (Pdet) at maximum flow (Pdet @ Qmax) rate on the Y axis, they created 3 zones representing obstructed, unobstructed, and equivocal micturition. The zone boundaries were created by a combination of empiric observations and theoretical

considerations. Conceptually, the Abrams-Griffiths nomogram does not permit a diagnosis of impaired detrusor contractility with or without coexisting BOO. The passive urethral resistance relation

(PURR) developed by Schafer [31,32] in 1983 constitutes a simplified

model of Griffith's URR. The PURR curve describes the relationship between pressure and flow during the period of lowest urethral resistance (i.e., during complete relaxation), and therefore defines the lowest urethral resistance during a single voiding event.

The importance of a minimum opening pressure in describing a collapsible

tube is considered. Outlet function is characterized by 2 simple parameters: the minimum opening pressure, reflecting

collapsibility of the tube, and the cross-sectional area of the flow-rate controlling zone, reflecting extensibility.[33] Therefore, the PURR curve is a method of assessing the presence or absence of BOO independent of inherent detrusor strength. The PURR was the first attempt to quantify relevant features of the voiding cycle describing the interplay of detrusor capability and bladder outlet resistance. Schafer subsequently modified the PURR by using a straight line instead of a parabolic curve.[34] Schafer divided this linear PURR (LinPURR) curve into 7 zones labeled 0 to VI corresponding to increasing grades of obstruction: grades 0 and 1, no obstruction; grade 2, equivocal or mild obstruction; grades 3 to 6, increasing severity of obstruction. The boundary between grades 2 and 3 corresponds to the boundary between equivocal and obstructed in the Abrams-Griffiths nomogram. The linear PURR

also allowed for the assessment of detrusor contractility independent of obstruction (strong, normal, weak, and very weak).

Finally in 1989, Griffiths and associates developed a single urethral resistance parameter, URA.[35] Using data from a mixed group of patients, they determined that obstruction is represented by URA values greater than 29 cm H2O. In the past 10 years, work has been done to simplify the diagnosis of BOO in men and to create a standardized method for diagnosis, based on the work of different authors described above. In 1997, the International Continence Society (ICS) introduced the provisional ICS nomogram, which is recommended for the diagnosis of obstruction in older men with LUTS suggestive of benign prostatic

obstruction (BPO).[36] This was based on extensive studies and concepts developed by Griffith, Abrams and Schafer. Lim and Abrams showed that patients were identically classified by the Abrams-Griffiths and Schafer nomograms and there was only a 6% discrepancy between these and the URA nomogram described by

Griffiths.[38] They also described the Abrams-Griffiths number derived from the slope of the dividing obstructed and equivocal

groups on the Abrams-Griffiths nomogram and the same line dividing the

obstructed (II) and slightly obstructed (III) on the Schafer nomogram. The Abrams-Griffiths number was later renamed the bladder outlet obstruction index (BOOI) and is represented by the equation: BOOI = Pdet @ Qmax – 2 Qmax.

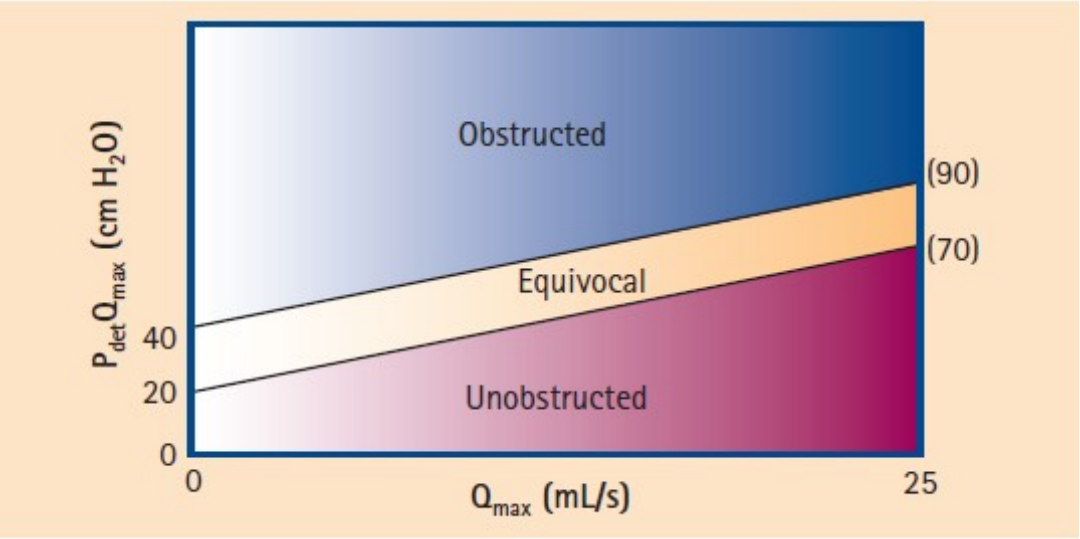

Figure 2: The ICS Nomogram. The patients are divided into 3 classes: unobstructed, obstructed and equivovocal based on the bladder outlet obstruction index (BOOI). From Abrams [35] ICS

International continence society; Pdet detrusor pressure; Qmax maximum flow rate.

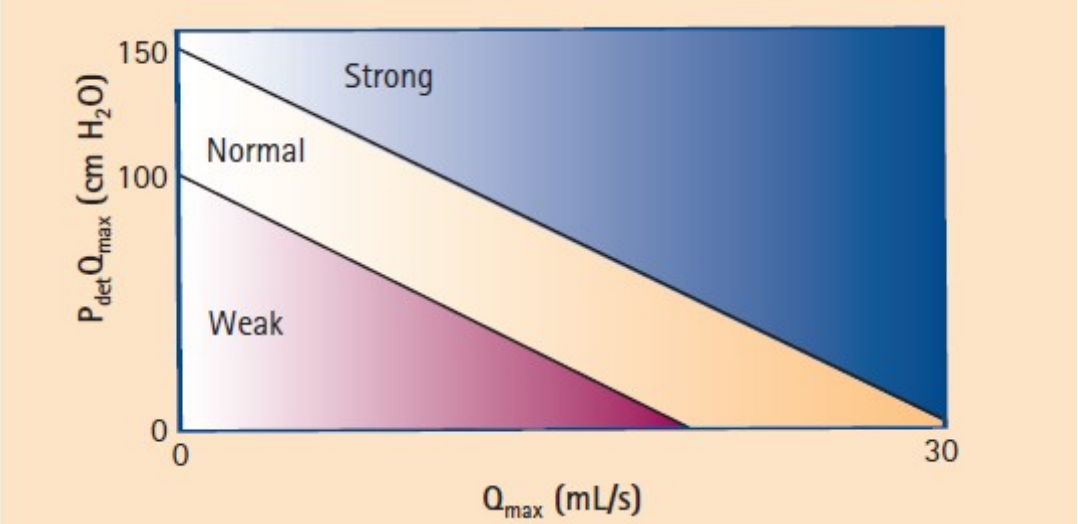

Figure 3: Bladder contractility nomogram. The patients are divided 3 classes; strong, normal and weak contractility based on Bladder Contractility Index (BCI). From Abrams [35] Pdet: detrusor pressure; Qmax Maximum flow rate

Using this ICS nomogram, men can be divided into obstructed, equivocal, and unobstructed according to their BOOI:

BOOI > 40 = obstructed;

BOOI 20-40 = equivocal; and

BOOI< 20 = unobstructed (Figure 2).

For purposes of standardization, this nomogram is recommended for use in older men with LUTS suggestive of BPO.

Significance of straining: Jensen et al[36] studied the frequency of abdominal straining during cystometry and pressure flow studies in men more than 50 yrs who presented with LUTS. Approximately 25-35% of men in this study either strained before the start of detrusor contraction, during the rise in detrusor pressure or during flow. Reynards et al[37] studied the prevalence of abdominal straining during free flow in patients with symptomatic BPH and it showed that approximately 25% of men strained during voiding. Garraway et al[38] in a study of the prevalence of symptoms of prostatic dysfunction in men aged 40-79 years in Scotland , found that only 0.4% of men said they strained to void and 11% said they occasionally strained to void. A survey of urinary symptoms in an unselected community based group of men in united

states[39]reported straining in 15% of men aged over 50 years. Two thirds of these subjects reported this symptom to be bothersome. In the study by Reynolds et al straining some or most of the time was reported by 13% of men and straining occasionally by 44%. A higher prevalence of symptoms is to be expected in men who specifically present with voiding problems. Straining to void is an even more common symptom in men undergoing TURP, prevalence rates of 30-40% being reported in two studies from United States.[40, 41] There are few reports relating the symptom of straining to void with objective evidence of straining during voiding. Jensen et al noted that half of the patients who claimed not to strain during voiding did actually show objective evidence of straining during micturition. Only a few studies have explored the relationship between abdominal straining and BOO. Susset et al commented that during voiding patients with obstruction usually required added pressure provided by straining. However the number in this study was small and no formal statistical

comparison of the prevalence of straining in the obstructed and the non obstructed groups. Further more the symptoms of straining were still present in 11 of 15 men following TURP in study by Jensen et al. Relief of obstruction therefore does not seem to remove the need to strain. Mefflan et al [42] and Christmas et al[43] studied the effect of abdominal straining on urinary flow rates in young men. In the former study, three men aged between 30 and 40 years performed multiple flow tests with and without abdominal straining. Straining caused a marked increase in flow rate. In a group of normal young men and women Christmas et al found that abdominal straining increased flow raters in men but not in women. Claridge et al[44] studied the effect of straining to void during free flows in a group of men with prostatic or bladder neck obstruction defined on basis of high voididng detrusor pressure and on cystoscopic findings. He found that in 29 of 31 patients abdominal straining had no effect on the flow rate. Infact in 10 men the flow rate fell with straining. He concluded that the fall in

flow rate indicated a rise in outflow resistance, probably as a result of external pressure on the intra abdominal part of the urethra. The flow rates remained unchanged or fell in most of the patients who Reynolds et al studied.

Patients & Methods:

Research Questions:

1. Is there an association between BOO and abdominal straining?
2. What is the incidence of straining during voiding in normal men?
3. Is there an Objective co-relation between the symptom of straining and abdominal pressure?

Inclusion Criteria for Cases:

Men more than 45 years of age with urodynamic proven obstruction as defined by the ICS criteria.

Inclusion criteria for controls: Normal Men more than 45 years who had no lower urinary tract symptoms

Exclusion Criteria for Cases:

1. Presence of Co morbidities like diabetes, hypertension, Ischaemic heart disease

2. Previous surgery of urethra, prostate or bladder.

3. Previous pelvic procedures likely to cause bladder denervation like Hysterectomy and Abdominoperineal excision of rectum.

4. Neurological diseases likely to influence the lower urinary tract 5. Overactive bladder

6. Bladder calculus.

7. Medications like anticholinergics, diuretics, antidepressants and antipsychotics.

8. Urinary tract infection

Calculation of Sample size: The sample size was calculated from a retrospective study comparing abdominal straining in those with BOO to those without

Methodology: The study was conducted in urology outpatient clinic of Christian medical college, Vellore. This was prospective case control study.

CASES: Men of age more than 45 years with LUTS attending the urology outpatient clinic were recruited for the study. They were asked to answer an international prostatic symptom score (IPSS) questionnaire (annexure 1). All the demographic and clinical data were recorded in the proforma (annexure 2). Evaluation of patients began with detailed history and examination done by the investigator. Estimation of serum creatinine, and urine microscopy was done for all. Urine culture and sensitivity was done for those who were subjected to urodynamics. Those who have positive culture with LUTS were excluded from the study. All subjects did a representative uroflowmetry. Post void residual urine was measured using abdominal ultrasound using prolate ellipsoid formula; Volume (V) in ml = Length x Height x transverse diameter x $\pi/6$ or 0.53.

Those with urinary free flow rate less than 10ml/sec underwent another free flow with abdominal pressure monitoring and urodynamic evaluation after written informed consent. The pressure-flow studies were done using medical measurement systems (MMS) UD 2000 equipment. Single dose of prophylactic antibiotic was given. Pretest residue was measured prior to urodynamic evaluation by placing two 6Fr infant feeding tubes. One of these tubes was used for filling as well as for intravesical pressure measurement. During cystometry in sitting posture, bladder was filled with physiological saline at 37ºC at a filling rate of 50ml/min. First sensation of bladder filling (ml), maximum cystometric capacity (ml), detrusor overactivity (presence or absence), incontinence (presence or absence), and compliance (cmH2O) were assessed during filling phase. Maximum urinary flow (Qmax, ml/sec), Maximum intravesical pressure on voiding (cmH2O), Voided volume (ml), events like abdominal straining were noted during voiding

phase. Abdominal pressure was recorded by using perforated rectal balloon catheter. Detrusor pressure was calculated by subtracting intra-abdominal pressure from intravesical pressure. Detrusor pressure at maximum urinary flow rate (Pdet at Qmax, cm H2O) was measured to evaluate detrusor contractility. Methods, definitions and units were appropriate to the standards recommended by the international continence society.

Controls:

Men more than 45 years without any LUTS were recruited as controls after written informed consent. All did a representative uroflowmetry. Those who had flow rates more than 25 ml/sec were included. These underwent another uroflowmetry with simultaneous measurement of abdominal pressure. This was measured using urodynamic perforated rectal balloon catheter .

Definitions:

1) **Urinary incontinence** was defined if the complaint of any involuntary leakage of urine was present in which stress urinary incontinence (the complaint of involuntary leakage on effort on exertion, or on sneezing or coughing), or urge urinary incontinence (the complaint of involuntary leakage accompanied by or immediately preceded by urgency) were noted.
2) **Normosensitive bladder** - Volume at first sensation of 150 -200ml
3) **Delayed first sensation –** Appreciation of first sensation of filling at volume ≥250ml or greater than 50% of maximal cystometric capacity.
4) **Detrusor overactivity –** Involuntary phasic increase in detrusor pressure that was difficult to control or could not be controlled by patient resulting in incontinence or voiding.
5) **Normal compliance –** Filling detrusor pressure of 5-20cmH2O in the absence of simultaneous detrusor contraction at maximum cystometric capacity.

6) **Normal maximum cystometric capacity –** Volume 350 to 600ml, at which there was bladder contraction that resulted in voiding or patient discomfort.

7) **Normal urinary flow rate –** Catheterized urine flow rate of more than 12ml/sec.

8) **Normal Pdet at Qmax -** > 10cm H2O or <40cmH2O during voiding with catheterized flow rate if more than 12ml/sec.

9) **Normal post void residue** – 50ml

10) **Hypocontractile detrusor** – Pdet at Qmax less than 10cmH2O or flat trace during voiding with or without abdominal straining.

11) **Bladder outflow obstruction** – Pdet at Qmax more than 40cmH2O with catheterized urine flow rate less than 12ml/sec. Abrams Griffith Number calculated as Pdet-2Qmax more than 40

Analysis

There were three groups for analysis

Group 1: Those with poor flow and proven BOO

Group 2: Those with poor flow but no obstruction

Group 3: Normal men without LUTS

All statistical analyses were performed using Statistical Package for the social Sciences (SPSS 11.0) for windows. Categorical data was presented using frequencies and percentage. Continuous data was described using mean ± standard deviation or median and range. Associations between categorical variables were assessed using chi-square test with yates' correction or fisher's exact test. Continuous variables were compared using student't' tests and Mann-Whitney tests were used for non-normal data. A p-value less than 0.05 was considered statistically significant.

Results

Patients included in final analysis: Total of 100 males were included in the study . Of these 60 were those who presented with LUTS and 40 who were asymptomatic. These were subdivided for analysis into three groups as above. The mean age of cases was 58 years and that of the controls was 53 years. Neither had any co morbidities.

Prevalence of Straining: Of those who presented with LUTS and had BOO on urodynamics (Group 1) approximately 40% strained on urodynamic studies as per the criteria defined for straining. In those who had LUTS but were not obstructed on urodynamics(group B) 58% strained. 30% of asymptomatic men also strained on free flow done with abdominal pressure monitoring by a rectal balloon. Comparison between these three groups was not statistically significant (p=0.12.)

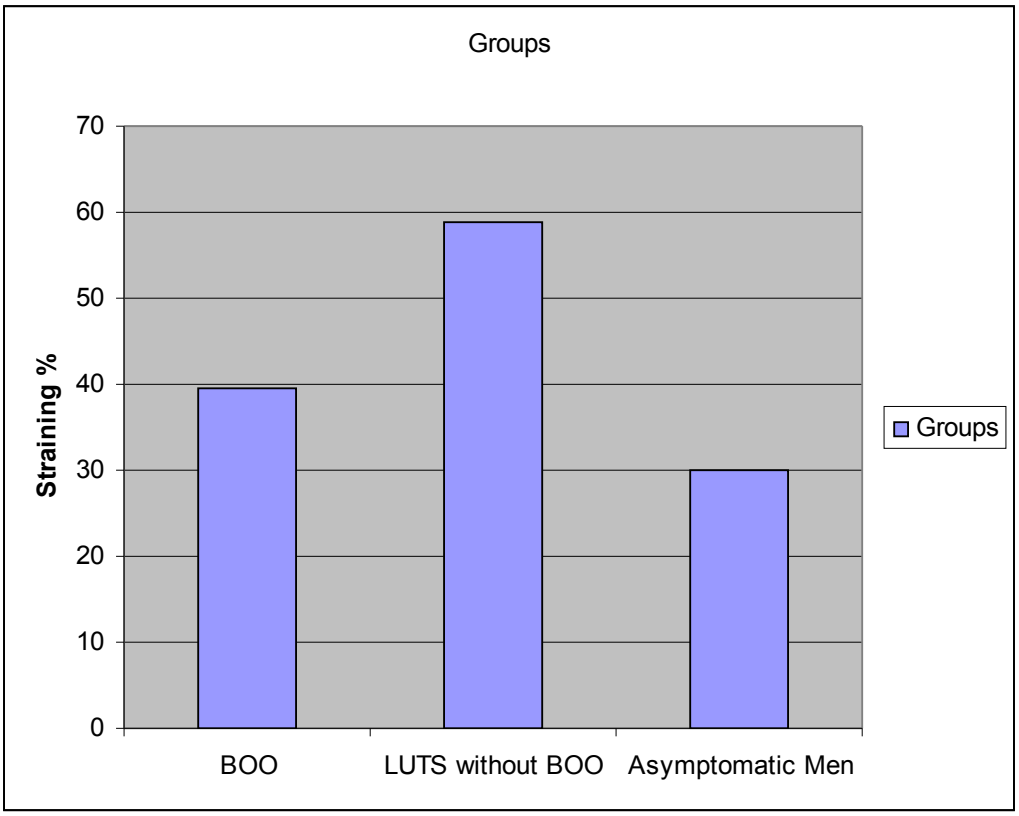

Figure 4: Comparison of objective evidence of straining on urodynamic/ free flow with abdominal pressure monitoring between the three groups

□

Straining as a symptom: 50% of those who presented with LUTS complained of straining to void. There was no statistical difference between the symptom of straining in those who had BOO and those who didn't. (P value 0.12). 52% of those who had no symptom of straining on the IPSS but had other LUTS had obstruction on urodynamics. Straining symptom therefore was a poor indicator of BOO.

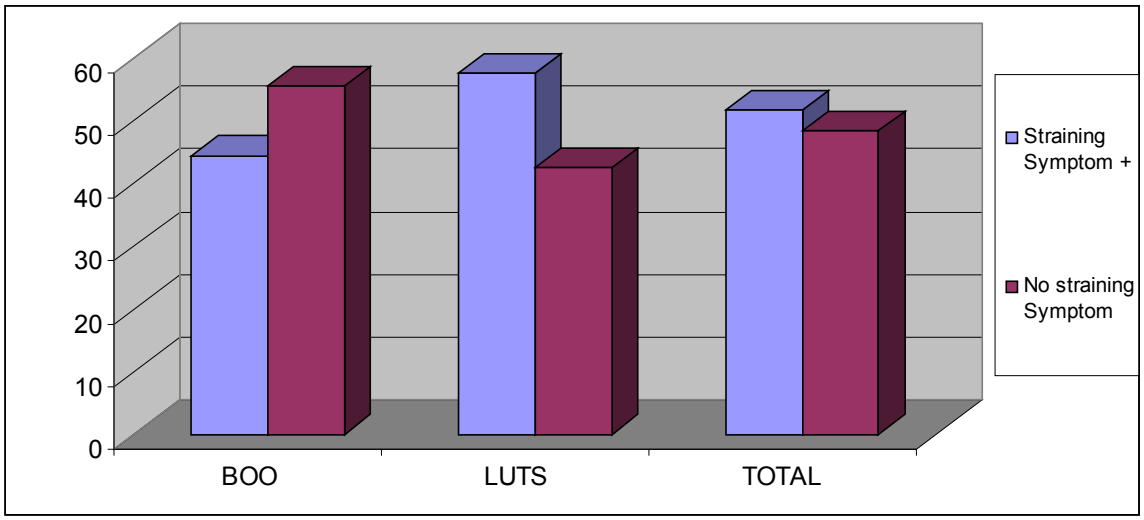

Figure 5: Comparison of the symptom of straining between those who had BOO to those who were not obstructed on urodynamics

Straining in Asymptomatic Men: 30% of asymptomatic men strained during free flow. Most of them commented that it was habitual and representative of their normal voiding pattern.

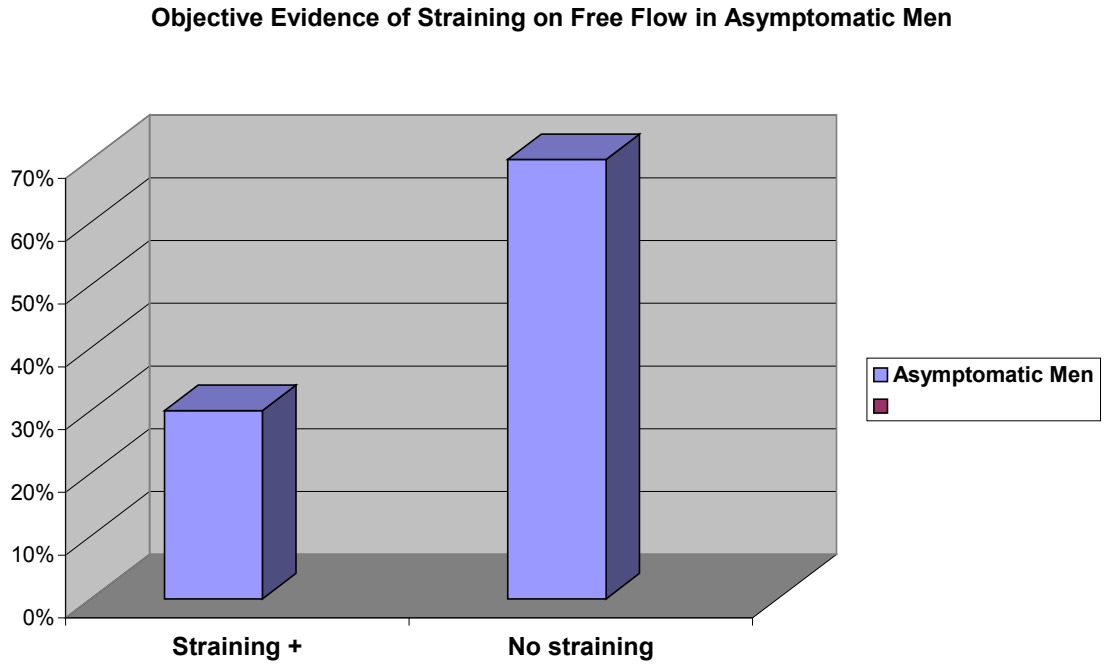

Figure 6: Straining in controls on free flow with rectal pressure monitoring.

Effect of Straining on the free flow rates: To ascertain the effect of straining on free flow rates a comparative analysis was done between those who strained and those who didn't in each of the groups. There was no statistically significant difference in mean, median, minimum or maximum flow rates in either group. Among cases the mean flow rate was 6 ml/sec and 6.2 ml/sec in those who strained and those who didn't respectively.

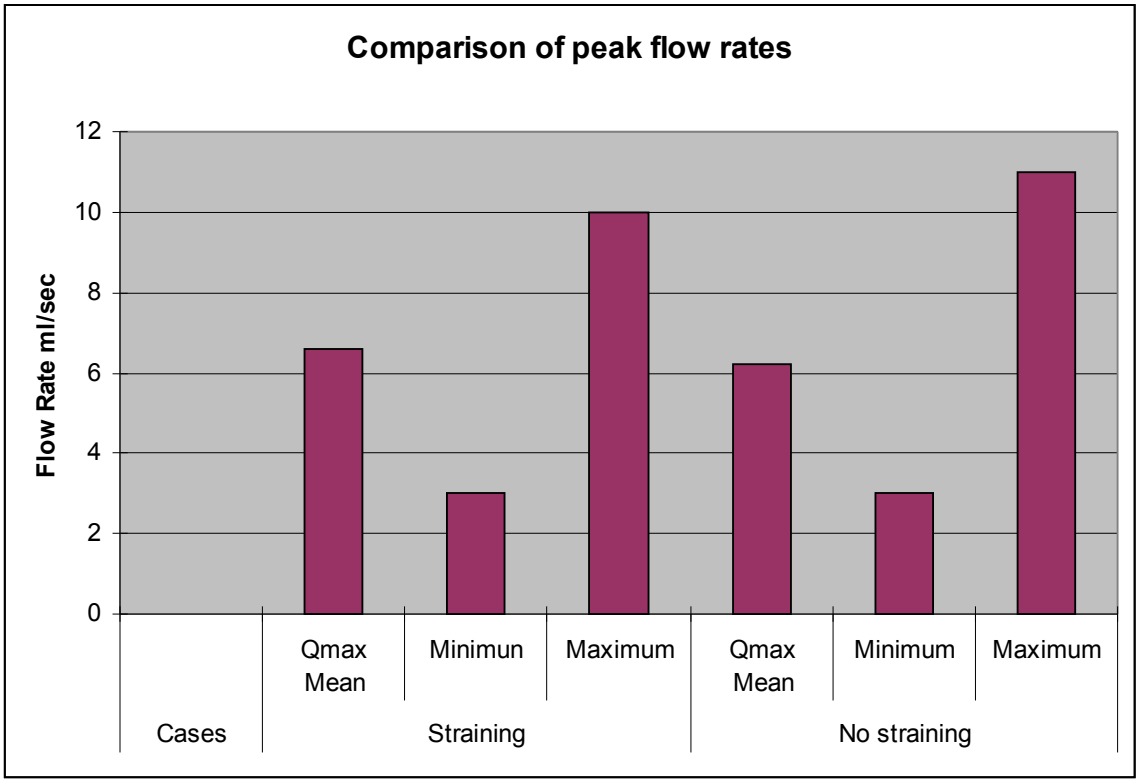

Figure: Comparison of flow rates in those with LUTS

Comparison among asymptomatic men did not reveal any statistical

difference in the mean, minimum or maximum flow rates among those who strained and those who didn't. (p value 0.311). An example of straining on free flow is shown in figure 7.

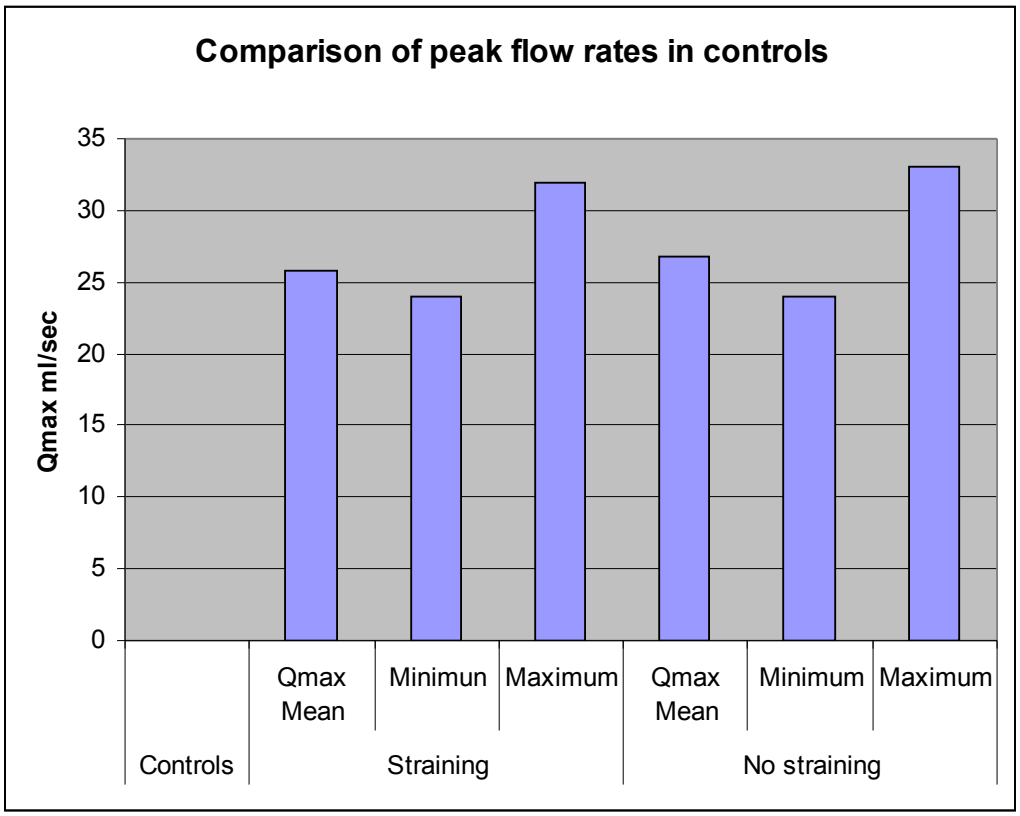

Figure 6: Comparison of flow rates in asymptomatic men

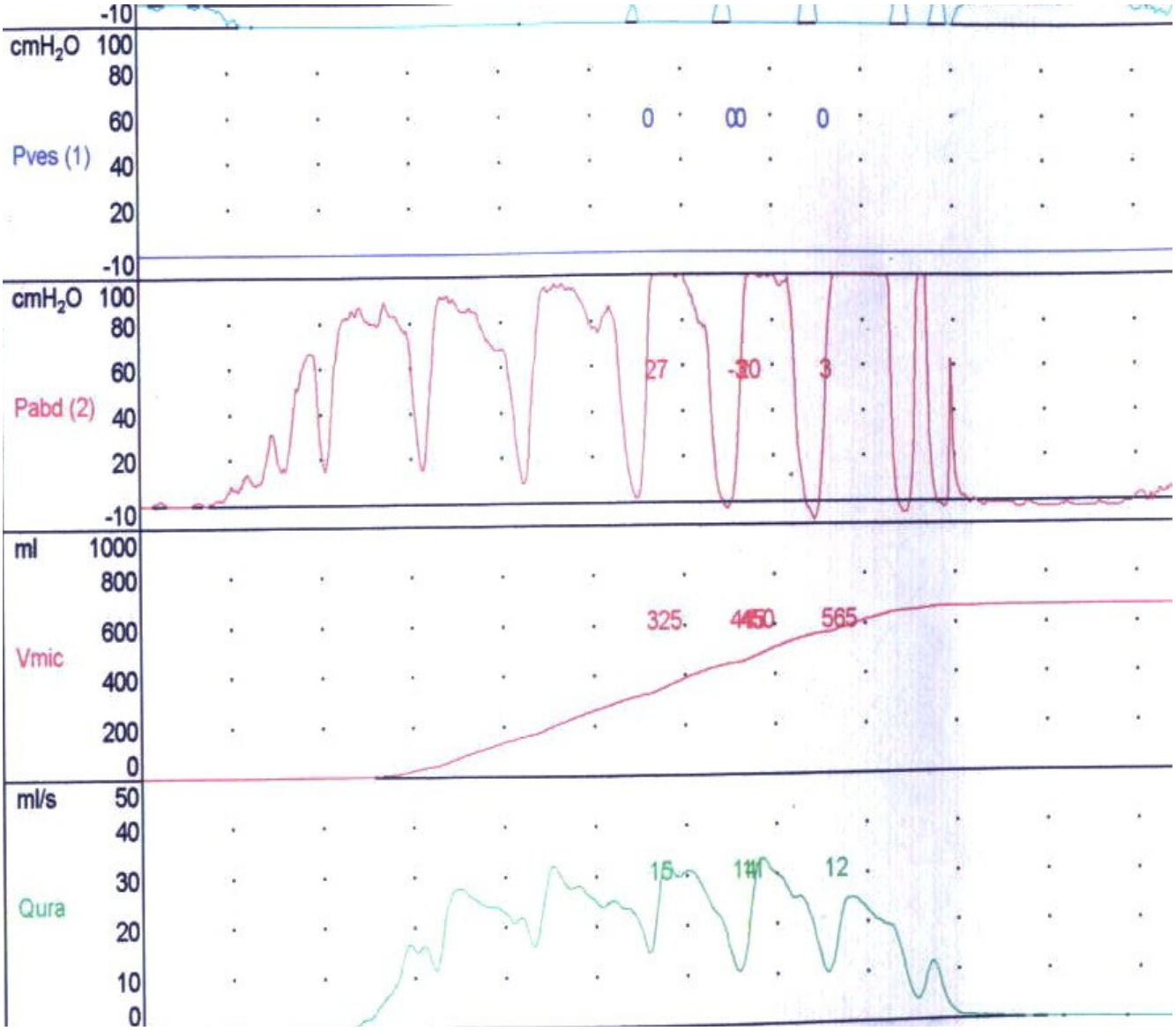

Figure 7: straining pattern on free flow with abdominal pressure monitoring in an asymptomatic male.

IPSS Scores: The mean IPPS score among the cases was 18. Apart from the symptom of straining other symptoms were analyzed in the cases. Among those who had BOO none of the storage or other voiding LUTS were statistically significant for BOO. Among those who had LUTS but no BOO on urodynamics none of the other symptoms had any statistically significant correlation (p value 0.16).

Discussion

The term ‘‘lower urinary tract symptoms’’ (LUTS) is an umbrella term that was introduced originally in 1994 to dissociate urinary symptoms in the male patients from any implied specific site of origin of symptoms, such as the prostate[19]. It is now recognized that LUTS is a global term that encompasses all urinary symptoms, including storage, voiding, and postmicturition symptoms. This terminology links well with the classification proposed by Wein[20], which suggested that disorders of micturition would be more elegantly characterized

as ‘‘failure to store’’ or ‘‘failure to empty.’’ In this context, it is important to acknowledge the fact that it has been known that symptoms do not relate to the underlying pathophysiology in many patients; indeed the phrase ‘‘the bladder is an unreliable witness’’ was coined to acknowledge this[45].Historically, voiding symptoms have been related to obstruction of the bladder outlet. The traditional association in men is with the prostate,

the so-called symptoms of ‘‘prostatism.’’ However, it is well recognized that voiding symptoms poorly correlate with underlying pathophysiology[46]. Similar symptoms can also be produced by any other form of obstruction, such as a urethral stricture or, conversely, by poor function of the lower urinary tract in circumstances in which there is impaired detrusor contractility. This has led to the recognition that, although LUTS may commonly be related to bladder outlet obstruction (BOO) as a result of benign prostatic obstruction (BPO), which is often associated with benign prostatic enlargement resulting from the histological condition of benign prostatic hyperplasia (BPH), this is not invariably the case. Failure to empty can be related either to an outlet obstruction or to detrusor underactivity of the bladder, or to a combination of both. Postmicturition symptoms, such as post void dribbling, occur in both sexes, but most often in men, in whom these symptoms are highly common, very troublesome, and cause significant interference with quality of life. Storage symptoms are currently largely encompassed by the term overactive bladder (OAB) syndrome, which is defined as urgency, frequency, nocturia, and urgency incontinence [47], and which is believed to

be correlated with underlying detrusor overactivity. These symptoms tend to be more bothersome than voiding symptoms, especially if they are associated with incontinence[48].The most recent international population-based survey, the EPIC study[49], was conducted in five countries using the 2002 ICS definitions for LUTS[38]. This survey assessed prevalence of OAB, urinary incontinence, and LUTS in more than 19 000 men and women. The data showed that there is a higher prevalence of storage (51.3%) versus voiding symptoms (25.7%) in men and all LUTS, including OAB, in addition to histologic BPH increase in prevalence as men age. The EPIC study demonstrated that the majority of men with voiding symptoms did not experience these in isolation but had either storage and/or postmicturition

symptoms as well. Specifically, among men with LUTS, 9% experienced both storage and voiding symptoms, whereas an additional 9% experienced storage, voiding, and postmicturition symptoms. It has also been shown that there is a far better correlation between storage LUTS and urodynamics than with Voiding LUTS. In our study however there was no statistically significant correlation between any of the 4 storage or the 3

voiding LUTS in the IPPS with the urodynamic findings. The EpiLUTS study, a cross-sectional, population representative Internet survey conducted in the United States, the United Kingdom, and Sweden to assess prevalence of LUTS, also found that both males and females who reported having voiding

symptoms were more likely to experience either storage or postmicturition symptoms, or both[50]. Data from the US study showed that 10.7% of the male population 40 yr and older had voiding symptoms, 10.1% experienced both voiding and storage symptoms, and 24.2% experienced voiding, storage, and postmicturition symptoms. In our study also most of the men who complained of voiding LUTS also had storage and post micturition symptoms.

Uroflowmetry remains a useful screening test in the evaluation of LUTS. It assesses the combination of detrusor force and outflow opening and, thus, gives an indirect indication of these aspects of bladder function. Flow rates must be interpreted together with the voided volume sine low volumes may give inaccurate flow-rate measurements. The most important parameter in men with LUTS is the maximum flow rate (Qmax);

additional information is gained by looking at the voiding time and the flow pattern. It is mandatory to have more than one flow-rate measurement, as they can be variable (depending on voided volume, diurnal variation). The voided volume should be>150ml. For patients with decreased flow rates who are suspected of BPO, urodynamic studies have shown that BOO was present in 88% of those with a Qmax <10 ml/s, in 57% of those with a Qmax of 10–14 ml/s, and in only 33% of those with a Qmax >15 ml/s [51]. Thus, a decreased flow rate implies a high likelihood of BOO due to BPO. Following this study by Abrams et al, a Qmax cut-off of 15 ml/s has been widely accepted as signifying BPO requiring treatment. To further increase the probability of detecting BOO we included those with flow rates of less than 10ml/sec as cases. Also a flow rate before the urodynamics was done to reduce the effect of intravesical lines on the maximum flow. Straining during voiding is one of the questions in the IPSS. There has been sparse literature on the correlation between the symptom and the objective documentation on urodynamics. In the previous studies by Jensen et al[36] and Reynard et al[37] 25-35% of men with LUTS strained during voiding. This is consistent with the findings in

our study in which 44% of men with urodynamic proven BOO strained. The incidence of straining was more in those with LUTS without obstruction (60%; 10/17) on pressure flow studies but it was not statistically significant. This could be due to the predominance of hypocontractile detrusors in this group. In this study the symptom of straining was present in overall 50% of patients with 44% of those with BOO complaining of straining. Reynard et al had reported straining some or most of the time in 13% and occasionally in 44% in those with BOO. A higher prevalence of symptoms is expected in men who specifically present with voiding problems. Indeed, straining to void is an even more common symptom in men undergoing transurethral resection of prostate (TURP) with prevalence rate of 35-40% being reported by Fowler and Bruskewitz et al.[40]

There have been few reports relating the symptom of straining to void with objective evidence of straining during voiding. Jensen et al noted that half of patients who claim not to strain did actually show objective evidence of straining during micturition. Thirteen of 61(21%) patients in the study by Reynard et al showed poor agreement between the symptom

of straining and objective evidence of its presence. In our study twenty seven (27/60) men with LUTS complained of straining to void. Only 12 of these (44%) had objective evidence of straining on pressure flow studies. Conversely of the 33 who did not complain of straining 19 actually strained. In the control group of normal men without any LUTS 30% strained. These observations suggest that a history of straining or not during urination may be unreliable and there is a poor correlation between the symptom of straining and its objective evidence. There was no consistent pattern in our study as to the timing of straining in the voiding curve. However in those without LUTS terminal straining was predominant though not statistically significant. This could be more due to habit of trying to expel the last few drops of urine. All of these men when retrospectively inquired also complained of straining to defecate suggesting a habitual pattern.

Few studies have looked at the relationship between abdominal straining and BOO. Jensen et al did not find any difference in detrusor pressures in men with and without straining. In our study there was no statistically significant difference in straining in those with BOO, those with LUTS

without BOO or in normal men. 44% of those with BOO and 30% of normal men strained during voiding. There was a poor correlation between straining to void and the presence of bladder outlet obstruction.

Straining to void in patients with BOO is believed to be an initiating cause of inguinal hernia. Most of the surgeons believe that relief of obstruction if possible be carried out prior to hernia repair. However as we have shown that the symptom of straining is unreliable and upto 50% of those who claim not to strain in fact do strain to void on objective assessment. Further more the straining pressures observed in patients who show the presence of straining are, in general, low compared to those observed with coughing. These findings in combination with the observation that straining does not cease after the relief of obstruction (Jensen et al) suggest that there is no support for the argument that evaluation to rule out BOO should be performed prior to inguinal hernia repair.

There was no difference in the mean or maximum flow rates between those who strained and those who didn't in all the 3 Groups. This is consistent with the study by Claridge et al who found that in 29 of 31patients abdominal straining had no effect on the flow rate. In their

study the rate in fact fell in 10 men. This was only seen in 4 men in our study who initially did not strain during free flow with abdominal pressure monitoring but later strained during urodynamics and was not significant. Our current understanding of the physiology of the urethra is that the urodynamic behavior of the bladder outlet is determined by the principles governing flow through distensible tubes. There is a flow controlling Zone, which in prostatic obstruction is located in the prostatic urethra, where it is under the influence of abdominal pressure. A rise in abdominal pressure probably results in an increase in outlet resistance thereby decreasing the flow. Another way to assess the effect of straining on flow rate could be by asking patients to whistle during voiding and then compare their flows.

Conclusions:

From this study it can be concluded that:

1) The relationship between the symptom of straining and the objective evidence of its presence is poor

2) Straining has a poor correlation with BOO and is not a sensitive measure of BOO

3) 30% of normal men strain habitually.

Limitations:

1) Small sample size

2) The effect straining has on the flow rate can be best assessed by having consecutive flows with subjects being asked to strain at different times during voiding. This can be incorporated in future studies.

3) The lack of associations between some of the parameters evaluated in this study might be due to the relatively small sample size and the consequent low power of the study. Studies with larger sample sizes can be performed in future to more accurately assess straining.

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Annexure: Worksheet for Controls. N=40,1 Present; 2 Absent

Age	H.No	free flow ml/sec	voided volume ml	PVR	flow with pressure	straining +/-	initial	at peak flow	continuous	intermittent
48	675133B	30	350	10	28	2				
51	404437D	32	400	20	30	1		1		
46	103507D	28	460	20	30	2				
50	410568D	25	520	30	26	2				
56	272972D	30	450	20	33	2				
47	401340D	26	500	20	27	2				
55	413305D	28	440	25	27	1				
48	567462C	25	400	15	25	1			1	
59	381381D	25	400	20	26	2				
68	382343D	24	380	20	22	1				1
57	383228D	25	600	30	26	1		1		
55	386103D	24	450	22	22	2				
57	388192D	25	360	20	24	2				
49	375628D	27	700	30	24	2				
52	386318D	25	300	10	26	2				
49	951812B	25	360	20	23	2				
55	893974C	26	550	20	25	1				
51	396548D	25	400	10	25	1				
52	396548D	29	570	20	28	2				
54	389564D	33	680	20	30	2				
66	395738D	26	440	20	25	2				
66	396399D	28	360	10	30	2				
55	176675D	24	340	20	27	2				
55	401128D	25	410	30	24	1			1	
48	080571D	26	300	20	24	2				
49	135497D	25	380	10	20	1	1			
46	771286	28	550	40	25	2				
54	455759C	24	600	10	25	1			1	
52	278952D	29	320	37	27	2				
49	392635D	25	300	46	24	1				
52	157705C	27	360	24	22	2				

	46	369550D	25	290	18	25	1
gh	67	558220A	25	300	16	20	2
	55	397575D	30	480	28	25	2
	48	405447D	26	380	22	22	2
	49	407173D	28	600	38	25	2
	50	225054D	26	300	22	22	2
	52	780564B	25	360	26	28	2
	49	411211D	25	300	10	27	2
	47	417001D	26	350	24	20	2

initial	at peak flow	continous	intermittent	terminal
	1			
				1
		1		
			1	
	1			
				1
				1
		1		
1				
		1		

1

WORKSHEET CASES N=60

	IPSS SCORE / 35	straining symptom	intensity /5	flow Qmax ml/sec	voided volume ml	PVR	Qmax with pressure	
563756	22	1	3	7	200	60	7	
365961d	24	1	2	7	250	40	6	
959924c	30	2		6	180	30	7	
366688 D	18	2		5	240	100	6	
368870 B	22	2		8	300	30	9	
092845 D	20	1	2	8	350	20	6	
375429d	23	1	4	5	158		4	
376159 D	18	2		9	250	30	11	
371729 D	20	1	4	8	200	60	9	
055778 D	14	2		9	170	150	10	
388265 D	20	1	1	4	200	40	5	
386486 D	23	2		8	340	20	10	
393868 D	12	1	2	8	560	10	7	
016226 C	20	1	2	3	200	40	4	
200691 D	26	2		4	380	60	3	
264925 D	21	1	1	8	260	20	6	
259532d	15	2		6	180	80	8	
239814 D	23	2		3	210	20	4	
370191 C	22	2		8	350	10	8	
271645 D	24	1	2	4	230	40	4	
266784d	22	1	3	5	180	50	6	
703435 A	18	1	2	5	300	30	4	
217568d	16	1	3	4	210	40	3	
240894 D	15	2		6	300	20	5	
325725 D	17	2		9	400	20	10	
388265 D	22	1	2	4	200	10	3	
333196 D	12	1	3	8	350	30	9	
187549 D	25	2		3	210	20	4	
301835 D	20	1	4	4	170	30	5	
939026 B	18	1	1	8	350	16	7	
299978 D	26	2		6	300	20	6	
263976 B	23	1	2	8	400	60	7	
295835 D	17	2		6	320	150	8	
298854 D	22	1	3	5	220	110	4	
283849 D	20	1	2	4	160	120	5	
234133 D	22	2		9	500	30	10	
068450								

straining +/-	initial	at peak flow	continous	intermittent	terminal	Pdet Qmax	Qmax	AG number	Straining +/-	initial	a

1			1			43	7	29	1
1			1			71	7	63	1
2						85	7	71	2
1					1	158	4	150	2
2						108	10	88	2
2						53	7	39	2
1			1			15	2	11	1
2						67	13	39	2
1				1		15	6	3	1
1	1					30	9	12	1
2						74	4	66	2
1				1		97	9	79	1
1			1			42	9	24	1
2						102	3	99	2
2						129	5	61	2
1	1					73	10	53	2
1		1				80	8	64	2
1					1	194	2	190	1
1					1	56	12	32	2
2						40	5	30	2
2						85	8	69	2
2						91	3	85	2
1			1			47	3	41	1
1		1				100	5	90	1
2						44	10	24	2
2						80	3	73	2
1			1			56	9	38	1
1		1				132	3	126	1
2						66	3	60	2
2						80	9	68	2
1				1		110	5	100	1
2						40	10	20	1
1			1			26	10	6	1
2						120	5	110	2
2						76	5	66	2
2						110	10	90	2
2						60	6	48	2
1			1			30	8	14	
1					1	183	7	169	1
2						100	5	90	2
2						102	4	94	2
2						68	5	58	2
2						77	5	67	2
1					1	104	8	96	1
2						83	6	71	2
1					1	110	5	100	1
2						129	7	115	2
2						60	10	40	2
2						60	12	46	2
2						95	8	79	2
1		1				83	4	75	1
2						60	3	53	2
2						80	4	72	2

1			1		102	2	98	1	
2					138	8	122	2	
2					88	3	82	2	
1				1	91	7	77	1	
1		1			78	8	62	1	1
2					88	5	78	2	
1	1				90	6	78	1	1

Straining +/-		initial	at peak flow	continous	intermittent	terminal	compliance	Instability
	1			1				
	1				1			
	2							
	2							
	2							1
	2							
	1			1				
	2							
	1			1			poor	
	1				1			
	2							
	1				1			
	1			1				
	2						poor	
	2							
	2							1
	2							
	1					1		
	2							
	2							
	2							
	2							
	1			1				
	1		1					
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	2							
	1				1			
	1				1			
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2					
1		1			
2					
2					
1			1		
2					
2					
1			1		
1	1				
2					
1	1				

Worksheet for Cases N=60
1: Present
2: Absent